52. (Previously Presented) The method of claim 51, wherein said TAO variant is selected from the group consisting of:

(a) amino acid residues 1-320 of TA02;

(b) amino acid residues 1-416 of TA02; and

(c) amino acid residues 15-285 of TA02.

53. (Previously Presented) The method of claim 49 or 50, wherein said modulator increases MAP kinase signal transduction.

54. (Previously Presented) The method of claim 49 or 50, wherein said modulator decreases MAP kinase signal transduction.

55. (Previously Presented) The method of claim 49 or 50, wherein said MEK3 or MEK6 activation is indicated by MEK3 or MEK6 phosphorylation.

56. (Previously Presented) The method of claim 55, wherein a decrease in MEK3 or MEK6 phosphorylation indicates a decrease in *MAP* kinase signal transduction.

57. (Previously Presented) The method of claim 55, wherein an increase in MEK3 or MEK6 phosphorylation indicates an increase in *MAP* kinase signal transduction.

58. (Previously Presented) The method of claim 49 or 50, wherein said agent is an antibody or antigen-binding fragment thereof.

59. (Previously Presented) The method of claim 58, wherein said antibody is a monoclonal antibody.

60. (Previously Presented) The method of claim 50, wherein said agent is an antisense polynucleotide or a ribozyme.

61. (Previously Presented) The method of claim 50, wherein said MEK3 or MEK6 activation is indicated by p38 activity.

62. (Previously Presented) The method of claim 61, wherein said p38 activity is indicated by p38 phosphorylation.

63. (Previously Presented) The method of claim 62, wherein a decrease in p38 phosphorylation indicates a decrease in MAP kinase signal transduction.

64. (Previously Presented) The method of claim 62, wherein an increase in p38 phosphorylation indicates an increase in MAP kinase signal transduction.

65. (Previously Presented) The method of claim 50, wherein said MEK3 or MEK6 activation is indicated by expression of a reporter gene under the control of a MEK3 or MEK6-dependent promoter.

66. (Previously Presented) The method of claim 65, wherein said MEK3 or MEK6-dependent promoter is ATF2.

67. (Previously Presented) The method of claim 49 or 50, wherein the TAO2 polypeptide or variant thereof is contacted with a MEK3 polypeptide

68. (Previously Presented) The method of claim 49 or 50, wherein the TAO2 polypeptide or variant thereof is contacted with a MEK6 polypeptide

69. (New) The method of claim 51, wherein said TAO variant comprises the catalytic domain.